L Number	Hits	Search Text	DB	Time stamp
1	5	ferrick-david-a.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/15 09:53
2	4	swift-susan-e.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/15 09:53
3	3	armstrong-randall.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/15 09:53
4	5	fox-bryan.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/15 09:54
5	23	retroviral same vector same promoter same reporter same inducible	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/15 09:57
6	200	retroviral same vector same promoter same reporter	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/15 09:57
-	7	wo adj "9214836"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/15 09:53
-	2	wo adj "9630515"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/14 10:13
-	1	de adj "4126414"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/14 10:13

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 NEWS  3  SEP 09   CA/CAplus records now contain indexing from 1907 to the
                   present
 NEWS  4  DEC 08   INPADOC: Legal Status data reloaded
 NEWS  5  SEP 29   DISSABS now available on STN
 NEWS  6  OCT 10   PCTFULL: Two new display fields added
 NEWS  7  OCT 21   BIOSIS file reloaded and enhanced
 NEWS  8  OCT 28   BIOSIS file segment of TOXCENTER reloaded and enhanced
 NEWS  9  NOV 24   MSDS-CCOHS file reloaded
 NEWS 10  DEC 08   CABA reloaded with left truncation
 NEWS 11  DEC 08   IMS file names changed
 NEWS 12  DEC 09   Experimental property data collected by CAS now available
                   in REGISTRY
 NEWS 13  DEC 09   STN Entry Date available for display in REGISTRY and CA/CAplus
 NEWS 14  DEC 17   DGENE: Two new display fields added
 NEWS 15  DEC 18   BIOTECHNO no longer updated
 NEWS 16  DEC 19   CROPU no longer updated; subscriber discount no longer
                   available
 NEWS 17  DEC 22   Additional INPI reactions and pre-1907 documents added to CAS
                   databases
 NEWS 18  DEC 22   IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields
 NEWS 19  DEC 22   ABI-INFORM now available on STN

 NEWS EXPRESS  DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT
               MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
               AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003
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=> s retroviral (p) vector (p) promoter (p) inducible (p) reporter (p) gene
L1          32 RETROVIRAL (P) VECTOR (P) PROMOTER (P) INDUCIBLE (P) REPORTER
               (P) GENE

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2          12 DUP REM L1 (20 DUPLICATES REMOVED)

=> d l2 total ibib kwic

L2   ANSWER 1 OF 12  CAPLUS  COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER:         2003:221443  CAPLUS
DOCUMENT NUMBER:          138:249719
TITLE:                    Expression of double-stranded RNA within cells using
                          viral vectors
INVENTOR(S):              Baltimore, David; Qin, Xiao-Feng; Lois-Caballe, Carlos
PATENT ASSIGNEE(S):       California Institute of Technology, USA
SOURCE:                   PCT Int. Appl., 51 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   4
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     WO 2003022052      A1   20030320       WO 2002-US29215  20020913
         W:  AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES,
             FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG,
             KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW,
             MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK,
             SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM,
             ZW, AM, AZ, BY
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG,
             CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR,
             NE, SN, TD, TG
     US 2003068821      A1   20030410       US 2002-243816   20020913
PRIORITY APPLN. INFO.:                     US 2001-322031P  P  20010913
                                           US 2002-347782P  P  20020109
                                           US 2002-389592P  P  20020618
                                           US 2002-406436P  P  20020827
REFERENCE COUNT:          13    THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB   The invention provides methods and compns. for the expression of small RNA
     mols. within a cell using a lentiviral vector.  The methods can
     be used to express double-stranded RNA complexes.  Small interfering RNA
```

(siRNA) can be expressed using the methods of the invention within a cell, which are capable of downregulating the expression of a target **gene** through RNA interference. A variety of cells can be treated according to the methods of the invention including embryos, embryonic stem cells, allowing for the generation of transgenic animals or animals constituted partly by the transduced cells that have a specific **gene** or a group of **genes** downregulated. In one aspect, the invention provides **retroviral** constructs for the expression of an RNA mol. or mols. within a cell. The constructs preferably comprise a nucleic acid having the R and U5 sequences from a 5' lentiviral long terminal repeat (LTR), a self-inactivating lentiviral 3' LTR, and a RNA polymerase III (pol III) **promoter**. The **retroviral** constructs preferably comprise an RNA coding region operably linked to the RNA Polymerase III **promoter**. The RNA coding region preferably comprises a DNA sequence that can serve as a template for the expression of a desired RNA mol. The RNA coding region can be immediately followed by a pol III terminator sequence which directs the accurate and efficient termination of RNA synthesis by pol III. In one embodiment, the RNA coding region encodes a self-complementary RNA mol. having a sense region, an antisense region and a loop region. A lentiviral construct contg. siRNA expression cassette designed to downregulate expression of the lacZ **gene** was composed of a pol II **promoter** and a small hairpin RNA coding region followed by a pol III terminator site. Transduction of cultured mammalian cells with retrovirus derived from the **retroviral** construct described in Example 1 was achieved (Figure 6). The **retroviral vector** encoding a small hairpin RNA mol. described in Example 1, was used to transfect cultured mammalian cells that express lacZ. The concd. virus prepns. were used to infect either mouse embryonic fibroblasts (MEF) or HEK293 cells which harbor both lacZ and firefly luciferase (Luc) **reporter genes**. A tet-**inducible** lacZ siRNA lentiviral **vector** was also prepd. as illustrated in Figure 8. The Tet-**inducible** siRNA expression cassette was able to regulate **gene** expression in response to Doxycycline treatment. Luciferase **gene**.

L2 ANSWER 2 OF 12 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 2002:370015 BIOSIS
DOCUMENT NUMBER: PREV200200370015
TITLE: CNS-specific and cAMP-inducible expression of fusion genes controlled by multiple human neuronal nitric oxide synthase promoters.
AUTHOR(S): Chen, Wei-Kang [Reprint author]; Wu, Kunyi; Hartt, Gregory [Reprint author]; Zhang, Deyu; Pierson, Shawn; Oberdick, John; Boris-Lawrie, Kathleen; Young, Anthony [Reprint author]
CORPORATE SOURCE: Molecular Cellular Developmental Biology Program/Neurobiotechnology Center, Ohio State University, 1060 Carmack Rd, Columbus, OH, 43210, USA
SOURCE: FASEB Journal, (March 22, 2002) Vol. 16, No. 5, pp. A952. print.
Meeting Info.: Annual Meeting of Professional Research Scientists on Experimental Biology. New Orleans, Louisiana, USA. April 20-24, 2002.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Jul 2002
Last Updated on STN: 3 Jul 2002

AB The human neuronal nitric oxide synthase **gene** (hNOS1) is transcribed by at least 10 distinct **promoters**. The complexity of this **gene** affords considerable flexibility with respect to spatial and temporal patterns of **gene** expression. In an effort to better understand these patterns, fusion **genes** under

transcriptional control by individual hNOS1 **promoters** have been expressed in transgenic mice and in cultured pheochromocytoma PC12 cells. **Promoter** complexes designated PR(5'1+5'2) and PR(5'3+5'4) are sufficient to target beta-galactosidase expression to sets of overlapping NOS1-positive neuronal structures in transgenic mice. Ecotopic expression in NOS1-negative structures is not observed. An additional **promoter**, designated PR(E2) and found within exon 2 of hNOS1, is also able to target CNS-specific expression of the beta-galactosidase **reporter**. The PR(5'1+5'2) and E2 **promoters** are cAMP-**inducible**, based on analysis of hNOS1 **promoter**-EGFP fusion **genes** introduced into PC12 cells via a **retroviral vector**. These data suggest that a cAMP-mediated signaling pathway might be important for modulation of human NOS1 **gene** expression via multiple **promoters** in the CNS.

L2 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:417175 CAPLUS
DOCUMENT NUMBER: 135:1218
TITLE: Dexamethasone inducible viral vector responding to host cell transcriptional activators and its therapeutic uses
INVENTOR(S): Galipeau, Jacques; Jaalouk, Diana E.; Eliopoulos, Nicoletta; Couture, Clement; Mader, Sylvie
PATENT ASSIGNEE(S): Centre for Translational Research In Cancer, Can.
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001040494	A1	20010607	WO 2000-CA1422	20001130
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1234047	A1	20020828	EP 2000-979305	20001130
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2003031650	A1	20030213	US 2002-161333	20020603

PRIORITY APPLN. INFO.: US 1999-168299P P 19991201
WO 2000-CA1422 W 20001130
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB The present invention relates to a drug **inducible vector** regulatable with a trans-activator native to a host, and to a transplantable autologous tissue capable of engrafting in a recipient without requiring toxic conditioning, for transgene delivery to a recipient. Current drug **inducible** host-**vector** systems are responsive to foreign non-eukaryotic transcriptional activators which are potentially immunogenic and affect the long-term survival and function thereof. The present invention provides a drug **inducible** expression **vector** comprising a transgene operably linked to a **reporter** and to an **inducible promoter** responsive to a transcriptional activator of a host when exposed to an effective amt. of a clin. acceptable drug. Such a **vector** may be introduced in a transplantable host derived from the recipient and capable

of engrafting in the recipient without requiring toxic conditioning. A **retroviral vector** using the GRE5 glucocorticoid-response element in the long terminal repeat to confer dexamethasone inducibility is described. Use of the **vector** to deliver a **gene** for erythropoietin under the very tight control of dexamethasone to rat bone marrow stroma is described.

L2 ANSWER 4 OF 12 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001238008 MEDLINE
DOCUMENT NUMBER: 21202012 PubMed ID: 11306481
TITLE: Gene therapy targeting for hepatocellular carcinoma: selective and enhanced suicide gene expression regulated by a hypoxia-inducible enhancer linked to a human alpha-fetoprotein promoter.
AUTHOR: Ido A; Uto H; Moriuchi A; Nagata K; Onaga Y; Onaga M; Hori T; Hirono S; Hayashi K; Tamaoki T; Tsubouchi H
CORPORATE SOURCE: Department of Internal Medicine II, Miyazaki Medical College, Kiyotake, Japan.
SOURCE: CANCER RESEARCH, (2001 Apr 1) 61 (7) 3016-21.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010517
Last Updated on STN: 20010517
Entered Medline: 20010503

AB We previously reported that the **retroviral vector** expressing the herpes simplex virus-thymidine kinase **gene** under the control of 0.3-kb human alpha-fetoprotein (AFP) **gene promoter** (AF0.3) provided the cytotoxicity to ganciclovir (GCV) in high-AFP-producing human hepatoma cells but not in low-AFP-producing cells. Therefore, specific enhancement of AFP **promoter** activity is likely to be required to induce enough cytotoxicity in low-AFP-producing hepatoma cells. In this study, we constructed a hybrid **promoter**, [HRE]AF, in which a 0.4-kb fragment of human vascular endothelial growth factor 5'-flanking sequences containing hypoxia-responsive element (HRE) was fused to AF0.3 **promoter**. By means of the **reporter gene** transfection assay, hypoxia-**inducible** transcriptions that were mediated by [HRE]AF **promoter** were detected in low- and non-AFP-producing human hepatoma cells, but not in nonhepatoma cells. When the herpes simplex virus-thymidine kinase **gene** controlled by [HRE]AF **promoter** was transduced into hepatoma and nonhepatoma cells by a **retroviral vector**, the exposure to 1% O2 induced GCV cytotoxicity specifically in the hepatoma cells. Moreover, in nude mice bearing solid tumor. . . xenografts, only the tumors consisting of the virus-infected hepatoma cells gradually disappeared by GCV administration. These results indicate that the hypoxia-**inducible** enhancer of the human vascular endothelial growth factor **gene**, which is directly linked to human AFP **promoter**, involves selective and enhanced tumoricidal activity in **gene** therapy for hepatocellular carcinoma.

L2 ANSWER 5 OF 12 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2001454463 MEDLINE
DOCUMENT NUMBER: 21390592 PubMed ID: 11499754
TITLE: Expression of exogenous tissue factor pathway inhibitor in vivo suppresses thrombus formation in injured rabbit carotid arteries.
AUTHOR: Golino P; Cirillo P; Calabro' P; Ragni M; D'Andrea D; Avvedimento E V; Vigorito F; Corcione N; Loffredo F; Chiariello M

CORPORATE SOURCE: Department of Internal Medicine, University of Naples Federico II, Italy.. golino@unina.it
SOURCE: JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY, (2001 Aug) 38 (2) 569-76.
Journal code: 8301365. ISSN: 0735-1097.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 20010814
Last Updated on STN: 20011008
Entered Medline: 20011004

AB . . . The aim of the present study was to test the hypothesis that retrovirus-mediated in vivo tissue factor pathway inhibitor (TFPI) **gene** transfer to the arterial wall would efficiently inhibit thrombosis without causing significant changes in systemic hemostatic variables. BACKGROUND: Acute coronary. . . formation. Tissue factor pathway inhibitor is a naturally occurring inhibitor of the extrinsic pathway. METHODS: In the present study, the **gene** coding for rabbit TFPI was inserted in a **retroviral vector** under control of a tetracycline-**inducible promoter**. Replication-defective, infectious, recombinant retroviruses were used to transfect rabbit carotid arteries with either TFPI or a **reporter gene**--green fluorescent protein (GFP). RESULTS: **Retroviral**-mediated arterial **gene** transfer of TFPI resulted in potent inhibition of intravascular thrombus formation in stenotic and injured rabbit carotid arteries, whereas transfection. . . systemic hemostatic variables (prothrombin time and partial thromboplastin time) were observed when thrombosis was inhibited. CONCLUSIONS: These data suggest that **retroviral**-mediated transfection of the arterial wall with TFPI might represent an attractive approach for the treatment of thrombotic disorders.

L2 ANSWER 6 OF 12 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2000214357 MEDLINE
DOCUMENT NUMBER: 20214357 PubMed ID: 10752683
TITLE: Eradication of murine mammary adenocarcinoma through HSVtk expression directed by the glucose-starvation inducible grp78 promoter.
AUTHOR: Chen X; Zhang D; Dennert G; Hung G; Lee A S
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, USC/Norris Comprehensive Cancer Center, University of Southlern California Keck School of Medicine, Los Angeles 90089-9176, USA.
CONTRACT NUMBER: CA27607 (NCI)
CA59318 (NCI)
SOURCE: BREAST CANCER RESEARCH AND TREATMENT, (2000 Jan) 59 (1) 81-90.
Journal code: 8111104. ISSN: 0167-6806.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 20000706
Last Updated on STN: 20000706
Entered Medline: 20000623

AB **Gene** therapy strategies employing the HSVtk/ganciclovir (GCV) suicide **gene** offer promising approaches towards the treatment of metastatic breast cancer. These include bystander effects on non-transduced tumor cells, lower systemic toxicity, and the possibility of inducing immunity against the tumor. Previously we have demonstrated the ability of the grp78 stress-**inducible promoter** to

stimulate expression of **reporter genes** within the tumor microenvironment. However, experimental evidence demonstrating the ability of this **promoter** to activate therapeutic agents within the breast cancer environment causing tumor eradication is needed prior to clinical trials. In this report, we test the efficacy of the grp78 **promoter** in a **retroviral** system to drive the expression of the HSVtk suicide **gene** in a murine mammary adenocarcinoma cell line (TSA) in syngeneic, immune-competent hosts. Our results show that under glucose-starvation conditions in vitro, the expression of HSVtk and GCV induced cell death are enhanced in tumor cells in which the HSVtk **gene** is driven by the internal grp78 **promoter** compared to cells in which the Moloney murine leukemia virus LTR drives HSVtk. In in vivo studies, in tumors in which the HSVtk **gene** is driven by the grp78 **promoter**, GCV treatment causes complete tumor eradication, whereas tumors persist when the HSVtk **gene** is driven by the **retroviral** LTR. Our study suggests that the grp78 **promoter** may be useful to enhance the effectivity of therapeutic agents within a breast tumor. In addition, it is shown that immune memory is induced in syngeneic, immune-competent hosts. This new **retroviral vector** might therefore be useful for breast cancer **gene** therapy.

L2 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:736905 CAPLUS
DOCUMENT NUMBER: 132:2779
TITLE: Methods and compositions for screening for modulators of IgE synthesis, secretion and switch rearrangement
INVENTOR(S): Ferrick, David A.; Swift, Susan E.; Armstrong, Randall; Fox, Bryan
PATENT ASSIGNEE(S): Rigel Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 81 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9958663	A1	19991118	WO 1999-US10497	19990512
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2328481	AA	19991118	CA 1999-2328481	19990512
AU 9939862	A1	19991129	AU 1999-39862	19990512
AU 765000	B2	20030904		
EP 1076695	A1	20010221	EP 1999-922992	19990512
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1998-76624 A 19980512
WO 1999-US10497 W 19990512
REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB The invention relates to methods and compns. useful in screening for modulators of IgE synthesis, secretion and switch rearrangement. The method comprises combining a bioactive agent and a cell comprising a fusion nucleic acid contg. an interleukin 4-**inducible** .epsilon. **promoter** and a **reporter gene**. The

promoter is then induced with interleukin 4 (or interleukin 13), and the presence or absence of the **reporter** protein is detected. Generally, the absence of the **reporter** protein indicates that the agent inhibits the interleukin 4-**inducible** .epsilon. **promoter**. The fusion nucleic acid may comprise an exogenous interleukin 4-**inducible** .epsilon. **promoter**, or an endogenous **inducible** .epsilon. **promoter**. Alternatively, fusion nucleic acids are provided comprising **promoters** of interest that are **inducible** (such as the IL-4 .epsilon. **promoter**), and hooked to a death **gene** that requires a death **gene** that requires a death ligand. Chimeric death receptors may comprise the extracellular domain of a ligand-activated multimerizing receptor and the endogeneous cytosolic domain of a death receptor **gene**, such as Fas or tumor necrosis factor. Preferred embodiments utilize **retroviral vectors** to introduce the candidate bioactive agents. In addn., cell lines CA-46 and MC-116 are provided for screening.

L2 ANSWER 8 OF 12 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 1999434746 MEDLINE
DOCUMENT NUMBER: 99434746 PubMed ID: 10505121
TITLE: Hematopoietic cytokine-inducible gene expression from retroviral vectors.
AUTHOR: Saylors R L; Stine K C; Derrick J
CORPORATE SOURCE: Department of Pediatrics, University of Arkansas for Medical Sciences, Little Rock 72202, USA.
SOURCE: GENE THERAPY, (1999 May) 6 (5) 944-6.
Journal code: 9421525. ISSN: 0969-7128.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199910
ENTRY DATE: Entered STN: 20000111
Last Updated on STN: 20000111
Entered Medline: 19991022

AB **Retroviral vectors** capable of cytokine-**inducible gene** expression will be useful for a number of **gene** therapy applications. We explored one mechanism whereby cytokine inducibility may be imparted to the **retroviral** U3 **promoter**/enhancer by utilizing the JAK-STAT signal transduction pathway that is activated by a number of hematopoietic cytokines. We used PCR mutagenesis. . . insertion of multimers of a STAT-binding oligonucleotide with the core sequence 5'-TTCCCGGAA. After insertion of the modified U3s into a **retroviral vector** expressing the luciferase **reporter gene** and transduction of the HepG2 cell line, luciferase expression was induced with recombinant human IFN-gamma. The level of induction reached a maximum of 9.9-fold higher than the uninduced **vector** when the Sp1-U3 contained four STAT oligos. When this optimal **vector** was compared with the wild-type and Sp1 **vectors**, respective values of 17.9- and 16.7-fold higher expression were achieved with IFN-gamma treatment. **Retroviral vectors** incorporating these cytokine-**inducible** U3s will be useful for **gene** therapy in a number of situations involving **gene** transfer to hematopoietic, hepatic and other cytokine-responsive cell types.

L2 ANSWER 9 OF 12 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 97374621 MEDLINE
DOCUMENT NUMBER: 97374621 PubMed ID: 9231070
TITLE: Employment of the mdr1 promoter for the chemotherapy-inducible expression of therapeutic genes in cancer gene therapy.
AUTHOR: Walther W; Wendt J; Stein U

CORPORATE SOURCE: Max-Delbriick-Center for Molecular Medicine, Berlin, Germany.
SOURCE: GENE THERAPY, (1997 Jun) 4 (6) 544-52.
Journal code: 9421525. ISSN: 0969-7128.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970825
Last Updated on STN: 19970825
Entered Medline: 19970812

AB Numerous approaches in **gene** therapy of human cancers are focused on the establishment of cell type specific or **inducible** expression **vectors** allowing the targeted and regulated expression of therapeutic **genes**. Various conditionally active **vectors** have been created carrying **promoters** responding to certain factors or therapeutic modalities (eg hormones, irradiation). The **promoter** of the multidrug resistance **gene** (mdr1) harbors such responsive elements and two of these elements have been related to drug responsiveness. In earlier studies we and others have characterized the mdr1 drug responsive-element in CAT **reporter** assays demonstrating its inducibility by MDR-associated drugs. To exploit this property, we linked the mdr1 **promoter** sequence to the human tumor necrosis factor alpha (TNF) cDNA in a **retroviral** **vector** and transduced the **vector** into human mammary and colon carcinoma cell lines. These cells were treated with various mdr1-associated drugs to induce TNF expression in vitro. We have shown that the mdr1 **promoter**-driven TNF expression is drug-**inducible** and that this induction is drug concentration and time dependent. The studies demonstrate the feasibility of the novel **vector** system for a chemotherapy-**inducible** expression of a chemosensitizing cytokine that is successful at enhancing cytotoxicity of drugs in cancer therapy.

L2 ANSWER 10 OF 12 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 95228047 MEDLINE
DOCUMENT NUMBER: 95228047 PubMed ID: 7712471
TITLE: Use of the stress-inducible grp78/BiP promoter in targeting high level gene expression in fibrosarcoma in vivo.
AUTHOR: Gazit G; Kane S E; Nichols P; Lee A S
CORPORATE SOURCE: Department of Biochemistry, University of Southern California School of Medicine, Los Angeles 90033-0800, USA.
CONTRACT NUMBER: CA27607 (NCI)
CA59308 (NCI)
T32 CA09569 (NCI)
SOURCE: CANCER RESEARCH, (1995 Apr 15) 55 (8) 1660-3.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199505
ENTRY DATE: Entered STN: 19950524
Last Updated on STN: 19950524
Entered Medline: 19950515

AB Current advances in human **gene** therapy open up new frontiers for molecular therapies of cancer. However, one major limitation in cancer **gene** therapy is the lack of a general tumor-specific **promoter** which allows stringent and high level expression of the therapeutic reagent in malignantly transformed but not normal tissues. Hallmark features. . . GRP78/BiP, a M(r) 78,000 endoplasmic reticulum-localized protein with chaperone and calcium-binding properties. We report here that a truncated rat grp78 **promoter** with most of

the distal basal elements removed can be utilized as a potent internal **promoter** in a **retroviral vector** to drive high level expression of a **reporter gene** in a murine fibrosarcoma model system. The stress-**inducible** grp78 **promoter** offers a novel approach for **gene** delivery systems targeting transcription in tumorigenic cells.

L2 ANSWER 11 OF 12 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 95035215 MEDLINE
DOCUMENT NUMBER: 95035215 PubMed ID: 7948133
TITLE: Expression from leukocyte integrin promoters in retroviral vectors.
AUTHOR: Bauer T R Jr; Osborne W R; Kwok W W; Hickstein D D
CORPORATE SOURCE: Medical Research Service, Seattle Veterans Affairs Medical Center, WA 98108.
CONTRACT NUMBER: DK38531 (NIDDK)
DK43530 (NIDDK)
SOURCE: HUMAN GENE THERAPY, (1994 Jun) 5 (6) 709-16.
Journal code: 9008950. ISSN: 1043-0342.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199411
ENTRY DATE: Entered STN: 19950110
Last Updated on STN: 19950110
Entered Medline: 19941129

AB Human **gene** therapy for diseases involving leukocytes would be facilitated by the identification of specific **promoter**/enhancer sequences capable of directing high levels of tissue and stage-specific expression of the requisite cDNA when used in a **retroviral vector**. We tested the **promoter** sequences from the leukocyte integrin CD11a (LFA-1), CD11b (Mac-1), and CD18 subunits in **retroviral vectors** to express a **reporter gene**, adenosine deaminase, in the human leukocyte cell lines K562 and HL-60. The leukocyte integrins are expressed in leukocytes, and they are **inducible** in HL-60 cells, a model system for myeloid differentiation. Although the leukocyte integrin **promoter**/enhancer sequences direct the expression of **reporter genes** in myeloid lineage cell lines in transient transfection assays, in these studies, the leukocyte integrin **promoters** direct low levels of **reporter gene** expression following **retroviral**-mediated transduction in K562 and HL-60 cells and selection of stable integrants. Treatment of HL-60 cells transduced with **retroviral vectors** containing the leukocyte integrin **promoters** with retinoic acid or phorbol myristate acetate results in less than a two-fold increase in **reporter gene** expression. These studies indicate that: (i) expression from the leukocyte integrin **promoters** from stable integrants in **retroviral vectors** does not parallel the results observed in transient transfection assays, and (ii) additional **promoter**/enhancer sequences will likely be required for these **promoters** to direct high levels of tissue and stage-specific expression in **retroviral vectors**.

L2 ANSWER 12 OF 12 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 8
ACCESSION NUMBER: 1992:77205 BIOSIS
DOCUMENT NUMBER: PREV199293045660; BA93:45660
TITLE: EFFICIENT TRANSFECTION OF CHICKEN CELLS BY LIPOFECTION AND INTRODUCTION OF TRANSFECTED BLASTODERMAL CELLS INTO THE EMBRYO.
AUTHOR(S): BRAZOLOT C L [Reprint author]; PETITTE J N; ETCHES R J; VERRINDER GIBBINS A M

CORPORATE SOURCE: DEP ANIMAL AND POULTRY SCI, UNIV GUELPH, GUELPH, ONTARIO, CANADA N1G 2W1
SOURCE: Molecular Reproduction and Development, (1991) Vol. 30, No. 4, pp. 304-312.
CODEN: MREDEE. ISSN: 1040-452X.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 2 Feb 1992
Last Updated on STN: 2 Feb 1992

AB. . . and primary chicken fibroblasts (PCFs) have been lipofected with a variety of lacZ constructs encoding Escherichia coli .beta.-galactosidase (.beta.-gal). A **reporter** construct (phspPTlacZpA) containing a mouse heat-shock protein 68 **gene** (hsp 68) **promoter** was used to establish conditions for efficient lipofection. The construct, in circular or linear plasmid form or as **reporter** sequences alone, was transferred efficiently by incubating cells for 3.5 h in a mixture of 6.2 .mu.g LipofectinTM (a cationic. . . preparation from Bethesda Research Laboratories) and 1.55-3.1 .mu.g DNA per mL DMEM. These lipofection conditions were used to transfer a **reporter** construct (pCBcMt-lacZ) containing a Zn2+ -**inducible** chicken metallothiionein (cMt) **promoter**, and constructs showing constitutive expression due to Rous sarcoma virus plus chicken .beta.-actin (pmiwZ) or cytomegalovirus (pMaori3) **promoters**. Endogenous chicken .beta.-gal and transferred bacterial .beta.-gal activity could be distinguished clearly by incubating the cells with the substrate, Xgal,. . . or 7.4, respectively. Expression of phspPTlacZpA in chicken cells did not appear to require specific induction of the mouse hsp68 **promoter**, whereas expression of pCBcMtlacZ required treatment of the cells for 6-12 h with 150 .mu.M ZnCl2. Bacterial .beta.-gal activity was. . . could represent transient or stable incorporation of the construct. Plated PCFs were lipofected as well, with stable incorporation of the **gene** construct indicated in 10% of postive events. Lipofected CBCs were injected into the subgerminal cavity of stage X (Eyal-Giladi and. . . mixture directly into the embryo. Refinement of these procedures could contribute to the developent of transgenic poultry, without reliance on **retroviral vectors** for DNA transmission or incorporation.

=> d his

(FILE 'HOME' ENTERED AT 10:03:23 ON 15 JAN 2004)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 10:03:39 ON 15 JAN 2004
L1 32 S RETROVIRAL (P) VECTOR (P) PROMOTER (P) INDUCIBLE (P) REPORTER
L2 12 DUP REM L1 (20 DUPLICATES REMOVED)

=> s inducible (p) epsilon (p) promoter (p) reporter (p) gene
L3 34 INDUCIBLE (P) EPSILON (P) PROMOTER (P) REPORTER (P) GENE

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 13 DUP REM L3 (21 DUPLICATES REMOVED)

=> d l4 total ibib kwic

L4 ANSWER 1 OF 13 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2003195817 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12515729
TITLE: Regulation of neutrophil and eosinophil secondary granule gene expression by transcription factors C/EBP epsilon and PU.1.
AUTHOR: Gombart Adrian F; Kwok Scott H; Anderson Karen L; Yamaguchi

Yuji; Torbett Bruce E; Koeffler H Phillip
CORPORATE SOURCE: Division of Hematology/Oncology, Cedars-Sinai Medical Center, Los Angeles, CA 90048, USA.. gombarta@csmc.edu
CONTRACT NUMBER: CA26038-20 (NCI)
DK54938 (NIDDK)
SOURCE: Blood, (2003 Apr 15) 101 (8) 3265-73.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200306
ENTRY DATE: Entered STN: 20030429
Last Updated on STN: 20030619
Entered Medline: 20030618

AB In the bone marrow of C/EBP **epsilon**(-/-) mice, expression of neutrophil secondary and tertiary granule mRNAs is absent for lactoferrin (LF), neutrophil gelatinase (NG), murine cathelin-like protein. . . it is severely reduced for neutrophil collagenase (NC) and neutrophil gelatinase-associated lipocalin (NGAL). In addition, the expression of eosinophil granule **genes**, major basic protein (MBP), and eosinophil peroxidase (EPX) is absent. These mice express C/EBP alpha, C/EBP beta, and C/EBP delta. . . levels similar to those of their wild-type counterparts, suggesting a lack of functional redundancy among the family in vivo. Stable **inducible** expression of C/EBP **epsilon** and C/EBP alpha in the murine fibroblast cell line NIH 3T3 activated expression of mRNAs for B9, MCLP, NC, and NGAL but not for LF. In transient transfections of C/EBP **epsilon** and C/EBP alpha, B9 was strongly induced with weaker induction of the other **genes**. C/EBP beta and C/EBP delta proteins weakly induced B9 expression, but C/EBP delta induced NC expression more efficiently than the other C/EBPs. The expression of MBP was inefficiently induced by C/EBP **epsilon** alone and weakly induced with C/EBP **epsilon** and GATA-1, but the addition of PU.1 resulted in a striking cooperative induction of MBP in NIH 3T3 cells. Mutation of a predicted PU.1 site in the human MBP **promoter**-luciferase **reporter** construct abrogated the response to PU.1. Gel-shift analysis demonstrated binding of PU.1 to this site. MBP and EPX mRNAs were. . . liver of PU.1(-/-) mice. Restitution of PU.1 protein expression restored MBP and EPX protein expression. This study demonstrates that C/EBP **epsilon** is essential and sufficient for the expression of a particular subset of neutrophil secondary granule **genes**. Furthermore, it indicates the importance of PU.1 in the cooperative activation of eosinophil granule **genes**.

L4 ANSWER 2 OF 13 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2001534489 MEDLINE
DOCUMENT NUMBER: 21465156 PubMed ID: 11580837
TITLE: The promoter of the operon encoding the F0F1 ATPase of Streptococcus pneumoniae is inducible by pH.
AUTHOR: Martin-Galiano A J; Ferrandiz M J; de la Campa A G
CORPORATE SOURCE: Unidad de Genetica Bacteriana (CSIC), Centro Nacional de Biologia Fundamental, Instituto de Salud Carlos III, 28220 Majadahonda, Madrid, Spain.
SOURCE: MOLECULAR MICROBIOLOGY, (2001 Sep) 41 (6) 1327-38.
Journal code: 8712028. ISSN: 0950-382X.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF368465
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011003
Last Updated on STN: 20020124

Entered Medline: 20011231

AB The **genes** encoding the subunits of the F0F1 membrane ATPase of Streptococcus pneumoniae were cloned and sequenced. The eight **genes**, transcribed to one mRNA, are organized in an operon encoding the c, a, b, delta, alpha, gamma, beta and **epsilon** subunits of 66, 238, 165, 178, 501, 292, 471 and 139 amino acid residues, respectively, that were expressed in an. . . the atp-specific mRNA, as shown by Northern blot and slot-blot analysis. Primer extension experiments and transcriptional fusions between the atp **promoter** and the **reporter** cat **gene** demonstrated that this pH-dependent increase in the mRNA was regulated at the level of initiation of transcription. Transcription of the operon occurs from a **promoter** with a consensus -35 box (TTGACA) and a -10 box (TACACT) that differs from the consensus (TATAAT). A point mutation at the -10 box of the **promoter** (change to TGCACT) avoided this increase, suggesting a role for this sequence in the pH-**inducible** regulation.

L4 ANSWER 3 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:250200 BIOSIS
DOCUMENT NUMBER: PREV200200250200
TITLE: PML modulates p73-dependent transcription and apoptosis.
AUTHOR(S): Bernassola, Francesca [Reprint author]; Salomoni, Paolo [Reprint author]; Melino, Gerry; Pandolfi, Pier Paolo [Reprint author]
CORPORATE SOURCE: Molecular Biology Program, Dept. of Pathology, Sloan-Kettering Institute for Cancer Research, MSKCC, New York, NY, USA
SOURCE: Blood, (November 16, 2001) Vol. 98, No. 11 Part 1, pp. 758a. print.
Meeting Info.: 43rd Annual Meeting of the American Society of Hematology, Part 1. Orlando, Florida, USA. December 07-11, 2001. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Apr 2002
Last Updated on STN: 24 Apr 2002

AB The promyelocytic leukemia (PML) **gene**, involved in the t(15;17) (q22;q12) translocation in acute promyelocytic leukemia (APL), acts in vivo as a cell growth and tumor suppressor. PML inactivation dramatically accelerates leukemia onset in PML-RARalpha transgenic mice and the PML **gene** is found mutated in a subset of human APL patients (see also Gurrieri et al.). PML has been implicated in. . . induction of programmed cell death. p73, a structural and functional homologue of the tumor suppressor protein p53, can transactivate the **promoters** of several p53-responsive **genes** that control apoptosis and cell cycle. Unlike p53, p73 gives rise to six distinct protein isoforms, alpha, beta, gamma, delta, **epsilon** and zeta, as a result of alternative splicing. We have previously shown that PML plays an important role as modulator. . . functions. We therefore examined whether PML would functionally interact with p73. In p53-null Saos-2 cell lines stably transfected with Tet-On **inducible** p73alpha, beta and gamma expression vectors, over-expression of PML potentiated the ability of p73 to transactivate the p21 and the bax **promoters** in a dose-dependent manner. Transfection of the p21- and bax-luciferase **reporter** constructs into wild-type and PML-/- mouse primary embryonic fibroblasts revealed a marked impairment of p73 transcriptional activity in PML deficient. . .

L4 ANSWER 4 OF 13 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 3
ACCESSION NUMBER: 2000314865 EMBASE

TITLE: Endothelin 1 transciption is controlled by nuclear factor-.kappa.B in AGE-stimulated cultured endothelial cells.
AUTHOR: Quehenberger P.; Bierhaus A.; Fasching P.; Muellner C.; Klevesath M.; Hong M.; Stier G.; Sattler M.; Schleicher E.; Speiser W.; Nawroth P.P.
CORPORATE SOURCE: Dr. P.P. Nawroth, Vascular Medicine Section, Department of Internal Medicine IV, University of Tubingen, Otfried Muller Str. 10, 72076 Tubingen, Germany
SOURCE: Diabetes, (2000) 49/9 (1561-1570).
Refs: 59
ISSN: 0012-1797 CODEN: DIAEAZ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB . . . with HbA(1c) levels <6% were just half the levels observed after incubation with erythrocytes from patients with HbA(1c) levels >8%. N(. **epsilon**.)-(carboxymethyl)lysine (CML)-containing protein isolated from patients' erythrocytes induced ET-1, and CML-containing protein-dependent ET-1 induction was blocked by the recombinant decoy peptide. . . and Northern blot) in a time- and dose-dependent manner. Transient transfection of BAECs with a chimeric construct containing the 5' **promoter** region of the ET-1 **gene** linked to a **reporter gene** confirmed that AGE induced ET-1 **promoter** activity. Electrophoretic mobility shift assay confirmed AGE-**inducible** binding of members of the nuclear factor-.kappa.b (NF-.kappa.B) family to a potential binding site at -2,090 bp. Binding was functionally. . . whereas overexpression of NF-.kappa.B p65 induced ET-1 even in the absence of AGEs. Thus, ET-1 transcription is controlled by the AGE-**inducible** redox-sensitive transcription factor NF-.kappa.B.

L4 ANSWER 5 OF 13 MEDLINE on STN
ACCESSION NUMBER: 2000456823 MEDLINE
DOCUMENT NUMBER: 20440419 PubMed ID: 10982895
TITLE: Engineering EGFP reporter constructs into a 200 kb human beta-globin BAC clone using GET Recombination.
AUTHOR: Orford M; Nefedov M; Vadolas J; Zaibak F; Williamson R; Ioannou P A
CORPORATE SOURCE: CAGT Research Group, The Murdoch Children's Research Institute, Royal Children's Hospital, Flemington Road, Parkville, Melbourne, Victoria 3052, Australia.
SOURCE: NUCLEIC ACIDS RESEARCH, (2000 Sep 15) 28 (18) E84.
Journal code: 0411011. ISSN: 1362-4962.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200009
ENTRY DATE: Entered STN: 20001005
Last Updated on STN: 20010521
Entered Medline: 20000925
AB GET Recombination, a simple **inducible** homologous recombination system for Escherichia coli, was used to target insertion of an EGFP cassette between the start and termination codons of the beta-globin **gene** in a 200 kb BAC clone. The high degree of homology between the **promoter** regions of the beta- and delta-globin **genes** also allowed the simultaneous generation of a delta-globin **reporter** construct with the deletion of 8.8 kb of intervening sequences. Both constructs expressed EGFP after transient transfection of MEL cells. Similarly, targeting of the EGFP cassette between the

promoter regions of the gamma-globin **genes** and the termination codon of the beta-globin **gene** enabled the generation of **reporter** constructs for both (A)gamma- and (G)gamma-globin **genes**, involving specific deletions of 24 and 29 kb of genomic sequence, respectively. Finally the EGFP cassette was also inserted between the **epsilon**- and beta-globin **genes**, with the simultaneous deletion of 44 kb of intervening sequence. The modified constructs were generated at high efficiency, illustrating the. . . lines with these globin constructs will facilitate the search for therapeutic agents that modify the expression of the individual globin **genes** in a physiologically relevant manner.

L4 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:736905 CAPLUS
DOCUMENT NUMBER: 132:2779
TITLE: Methods and compositions for screening for modulators of IgE synthesis, secretion and switch rearrangement
INVENTOR(S): Ferrick, David A.; Swift, Susan E.; Armstrong, Randall; Fox, Bryan
PATENT ASSIGNEE(S): Rigel Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 81 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9958663	A1	19991118	WO 1999-US10497	19990512
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2328481	AA	19991118	CA 1999-2328481	19990512
AU 9939862	A1	19991129	AU 1999-39862	19990512
AU 765000	B2	20030904		
EP 1076695	A1	20010221	EP 1999-922992	19990512
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1998-76624 A 19980512
WO 1999-US10497 W 19990512
REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB The invention relates to methods and compns. useful in screening for modulators of IgE synthesis, secretion and switch rearrangement. The method comprises combining a bioactive agent and a cell comprising a fusion nucleic acid contg. an interleukin 4-**inducible** . **epsilon**. **promoter** and a **reporter gene** . The **promoter** is then induced with interleukin 4 (or interleukin 13), and the presence or absence of the **reporter** protein is detected. Generally, the absence of the **reporter** protein indicates that the agent inhibits the interleukin 4-**inducible** .**epsilon**. **promoter**. The fusion nucleic acid may comprise an exogenous interleukin 4-**inducible** . **epsilon**. **promoter**, or an endogenous **inducible** .**epsilon**. **promoter**. Alternatively, fusion nucleic acids are provided comprising **promoters** of interest that are **inducible** (such as the IL-4 .**epsilon**. **promoter**

), and hooked to a death **gene** that requires a death **gene** that requires a death ligand. Chimeric death receptors may comprise the extracellular domain of a ligand-activated multimerizing receptor and the endogeneous cytosolic domain of a death receptor **gene**, such as Fas or tumor necrosis factor. Preferred embodiments utilize retroviral vectors to introduce the candidate bioactive agents. In addn., cell lines CA-46 and MC-116 are provided for screening.

L4 ANSWER 7 OF 13 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 1998447473 MEDLINE
DOCUMENT NUMBER: 98447473 PubMed ID: 9776578
TITLE: A highly sensitive and specific assay using a novel human growth hormone cDNA **reporter gene** regulated by the human interleukin-4 **inducible** germline **epsilon** transcript **promoter**.
AUTHOR: Jenh C H; Cox M A; Lundell D; Narula S K; Zavodny P J
CORPORATE SOURCE: Department of Immunology, Schering-Plough Research Institute, Kenilworth, NJ 07033, USA.. chung-her.jenh@spcorp.com
SOURCE: JOURNAL OF IMMUNOLOGICAL METHODS, (1998 Aug 1) 217 (1-2) 87-95.
Journal code: 1305440. ISSN: 0022-1759.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981027

TI A highly sensitive and specific assay using a novel human growth hormone cDNA **reporter gene** regulated by the human interleukin-4 **inducible** germline **epsilon** transcript **promoter**.

AB We have successfully developed a highly sensitive and specific assay system for human interleukin-4 (IL-4) regulated **gene** expression. It is based on a human Jijoye cell line with the germline **epsilon** transcript **promoter** joined to the human growth hormone (hGH) cDNA. The germline **epsilon** transcript **promoter** is responsive to IL-4 and involved in immunoglobulin heavy chain class switching. We cloned hGH complementary DNA (cDNA) as the **reporter gene** instead of using conventional hGH genomic DNA which failed to generate any IL-4 **inducible** clone in human Jijoye cells. The two IL-4 **inducible** cell lines with the hGH cDNA **reporter** show high signal/noise ratio for IL-4-mediated induction (60-90 fold). The response to IL-4 is dose-dependent with ED50 of 10 pM.. . . factors, as well as mouse IL-4. The mutant hIL-4 antagonist hIL-4.Y124D inhibits the induction mediated by native hIL-4. These IL-4 **inducible** cell lines provide a sensitive, specific assay system to study IL-4-regulated **gene** expression, and in particular the regulation of the germline **epsilon promoter**.

L4 ANSWER 8 OF 13 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 97184462 MEDLINE
DOCUMENT NUMBER: 97184462 PubMed ID: 9032264
TITLE: Cloning of the novel human myeloid-cell-specific C/EBP-epsilon transcription factor.
AUTHOR: Chumakov A M; Grillier I; Chumakova E; Chih D; Slater J; Koeffler H P
CORPORATE SOURCE: Department of Medicine, Cedars-Sinai Medical Center, UCLA School of Medicine, Los Angeles, California 90048, USA.
CONTRACT NUMBER: CA42710 (NCI)
DK41936 (NIDDK)
DK42792 (NIDDK)

SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (1997 Mar) 17 (3) 1375-86.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U80982
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970327
Last Updated on STN: 20030225
Entered Medline: 19970314

AB Chicken NF-M transcription factor, in cooperation with either c-Myb or v-Myb, is active in the combinatorial activation of myeloid-cell-specific **genes** in heterologous cell types, such as embryonic fibroblasts. In humans, similar effects were observed with homologous members of the CCAAT/enhancer-binding protein (C/EBP) family of transcriptional regulators, especially the human homolog of chicken NF-M, C/EBP-beta (NF-IL6). However, the NF-IL6 **gene** is expressed in a variety of nonmyeloid cell types and is strongly **inducible** in response to inflammatory stimuli, making it an unlikely candidate to have an exclusive role as a combinatorial differentiation switch. . . the DNA-binding domains of highly homologous members of the C/EBP family of transcriptional regulators, we have cloned a novel human **gene** encoding a member of the C/EBP **gene** family, identified as the human homolog of CRP1, C/EBP-**epsilon**. A 1.2-kb cDNA encoding full-length human C/EBP-**epsilon** was cloned from a promyelocyte-late myeloblast-derived lambda gt11 library. Molecular analysis of the cDNA and genomic clones indicated the presence. . . encoding a protein with an apparent molecular mass of 32 kDa and a pI of 9.5. Primer extension analysis of C/EBP-**epsilon** mRNA detected a single major transcription start site approximately 200 bp upstream of the start codon. The putative **promoter** area is similar to those of several other myeloid-cell-specific **genes** in that it contains no TATAAA box but has a number of purine-rich stretches with multiple sites for the factors. . . expression pattern, with the strongest expression occurring in promyelocyte and late-myeloblast-like cell lines. Western blot and immunoprecipitation studies using rabbit anti-C/EBP-**epsilon** antibodies raised against the N-terminal portion of C/EBP-**epsilon** (amino acids 1 to 115) showed that C/EBP-**epsilon** is a 32-kDa nuclear phosphoprotein. The human C/EBP-**epsilon** protein exhibited strong and specific binding to double-stranded DNA containing consensus C/EBP sites. Cotransfection of the C/EBP-**epsilon** sense and antisense expression constructs together with chloramphenicol acetyltransferase **reporter** vectors containing myeloid-cell-specific c-mim and human myeloperoxidase **promoters** suggested a role for C/EBP-**epsilon** transcription factor in the regulation of a subset of myeloid-cell-specific **genes**. Transient tranfection of a promyelocyte cell line (NB4) with a C/EBP-**epsilon** expression plasmid increased cell growth by sevenfold, while antisense C/EBP-**epsilon** caused a fivefold decrease in clonal growth of these cells.

L4 ANSWER 9 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1995:766935 CAPLUS
DOCUMENT NUMBER: 123:167378
TITLE: Interleukin-2 promoter activity in Epstein-Barr virus-transformed B lymphocytes is controlled by nuclear factor-.chi.B
AUTHOR(S): Mouzaki, Athanasia; Serfling, Edgar; Zubler, Rudolf H.
CORPORATE SOURCE: Division ofd Hematology, Cantonal Univ., Geneva, Switz.
SOURCE: European Journal of Immunology (1995), 25(8), 2177-82
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: VCH

DOCUMENT TYPE: Journal
LANGUAGE: English
AB The regulation of interleukin (IL)-2 **gene** expression has been investigated mainly in T lymphocytes, the predominant producers of IL-2. However, B cells can also synthesize IL-2. In the present study we analyzed the control of IL-2 **promoter** activity in Epstein-Barr virus (EBV)-transformed B cell clones which are capable of secreting IL-2 at a low level after stimulation with phorbol 12-myristate 13-acetate and the Ca2+ ionophore ionomycin. Transient transfections using **reporter** constructs with multiples of transcription factor binding sites from the IL-2 **promoter** [distal nuclear factor (NF)-AT, proximal NF-AT, AP-1/Octamer (UPS) or NF-.chi.B (TCEd) sites] were performed. In EBV-transformed B clones, the .chi.B site exerted the strongest **inducible** activity; the NF-AT binding sites showed either no or only weak activity compared to Jurkat T cells. An IL-2 **promoter** b.**epsilon**.aring a defective NF-.chi.B site was completely inactive in EBV-B cells, while it still had activity in Jurkat T cells. In seven EBV-B cell clones or lines differing in their capacity to secrete IL-2, the activity of the Il-2 **promoter** correlated well with the status of IL-2 secretion. Similarly, a human immunodeficiency virus **promoter**, whose activity is controlled through .chi.B factors, was found to be active in the IL-2-producing EBV-B cells, but inactive in the non-IL-2-producing cells. Electrophoretic mobility shift assays using protein exts. from EBV-B cells and the IL-2 NF-.chi.B probe revealed the constitutive generation of .chi.B complexes in IL-2-secreting cells consisting mainly of heterodimeric p50/p65 complexes. A weaker .chi.B complex formation and faster-migrating complexes were detected in non-IL-2-secreting cells. These results demonstrate that the IL-2 NF-.chi.B site is indispensable for the activity of the IL-2 **promoter** in EBV-transformed B cells, whereas other transcription factors appear to be less important for IL-2 expression in these cells.

L4 ANSWER 10 OF 13 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 94194051 MEDLINE
DOCUMENT NUMBER: 94194051 PubMed ID: 8144891
TITLE: The transcription factor BSAP (NF-HB) is essential for immunoglobulin germ-line epsilon transcription.
AUTHOR: Liao F; Birshtein B K; Busslinger M; Rothman P
CORPORATE SOURCE: Department of Cell Biology, Albert Einstein College of Medicine, Bronx, NY 10461.
CONTRACT NUMBER: AI13509 (NIAID)
PC30CA13330 (NCI)
SOURCE: JOURNAL OF IMMUNOLOGY, (1994 Mar 15) 152 (6) 2904-11.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199405
ENTRY DATE: Entered STN: 19940511
Last Updated on STN: 20000303
Entered Medline: 19940504
AB . . . and certain B-lineage cell lines with mitogen (LPS) and the lymphokine IL-4 has been shown to induce expression of germ-line **epsilon** transcripts (l **epsilon** transcripts) and class switching to the C **epsilon gene**. Three protein complexes, one of which (complex 3) is constitutively expressed, have been shown to bind to a 179-base pair LPS/IL-4-responsive l **epsilon promoter** (Rothman, P., S. C. Li, B. Gorham, L. Glimcher, F. W. Alt, and M. Boothby. 1991. Mol. Cell. Biol. 11:5551). Complex 3 is indispensable for this **inducible promoter** activity.
In this report, we have used electrophoretic mobility shift assays (EMSA) to demonstrate that the early B cell-specific transcription factor (BSAP)

is involved in the formation of complex 3. In addition, BSAP is implicated functionally in l **epsilon** transcription because a BSAP binding site either from a sea urchin histone **promoter** (H2A-2.2) or from 5' of murine immunoglobulin S gamma 2a can substitute for the **epsilon**-associated site (**epsilon**(foot), as assayed by transient transfection assays of the l **epsilon**:CAT **reporter** constructs into the M12.4.1 B cell line. Like the sea urchin histone BSAP site, the complex 3 binding site (**epsilon** (foot)) functions as an upstream **promoter** element when assayed in the OVEC vector. These results indicate that BSAP is an essential protein required for LPS/IL-4 induction of the l **epsilon** **promoter**. In addition, experiments showing that a BSAP binding site from 5' of S gamma 2a also functions as an upstream **promoter** element in OVEC suggest a potential role for BSAP in regulation of the IgG2a isotype.

L4 ANSWER 11 OF 13 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 95001216 MEDLINE
DOCUMENT NUMBER: 95001216 PubMed ID: 7917786
TITLE: Activation of c-fos expression by transforming Ha-ras in HC11 mouse mammary epithelial cells is PKC-dependent and mediated by the serum response element.
AUTHOR: Uberall F; Kampfer S; Doppler W; Grunicke H H
CORPORATE SOURCE: Institute of Medical Chemistry and Biochemistry, University of Innsbruck, Austria.
SOURCE: CELLULAR SIGNALLING, (1994 Mar) 6 (3) 285-97.
Journal code: 8904683. ISSN: 0898-6568.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941222
Last Updated on STN: 20000303
Entered Medline: 19941028

AB . . . epithelial cells was investigated with regard to controversial data concerning the role of protein kinase C (PKC) and the required **promoter** elements of the fos **gene**. HC11 cells carrying a glucocorticoid-**inducible** Ha-ras (val12) construct were transfected with a chloramphenicol acetyltransferase (CAT) **reporter** **gene** under the control of a human fos **promoter** which includes the serum response element (SRE), the adjacent c-fos AP-1 site (FAP) and the cAMP response element (CRE). Induction of the Ha-ras **gene** by dexamethasone lead to a transactivation of expression of the transfected fos **promoter** construct which was inhibited by the PKC inhibitor BM41440 and abrogated in PKC-'depleted' cells. A similar transactivation was observed when the fos **promoter** construct was co-transfected with a constitutively active ras expression vector. Again, this effect was depressed by the PKC inhibitor and. . . by long-term exposure to 12-O-tetradecanoylphorbol-13-acetate. This procedure was shown to deplete cells of PKC alpha and to reduce significantly PKC **epsilon**. Long-term exposure to bryostatin 1 selectively depletes PKC alpha. Depletion of PKC alpha by bryostatin 1 does not reduce the transcriptional activation of the SRE-FAP-TK-CAT (TK: thymidine kinase) construct by Ha-ras. In order to delineate the **promoter** elements mediating the transcriptional activation, constructs which lack the FAP and the CRE sites but contain an intact SRE were. . . (val12). It is concluded that in HC11 cells, transforming Ha-ras activates c-fos expression in a PKC-dependent manner, presumably implying PKC **epsilon**, and that the SRE is sufficient to mediate transcriptional activation.

L4 ANSWER 12 OF 13 MEDLINE on STN
ACCESSION NUMBER: 95028757 MEDLINE

DOCUMENT NUMBER: 95028757 PubMed ID: 7942278
TITLE: Role of protein kinase C in ras-mediated fos-expression.
AUTHOR: Uberall F; Kampfer S; Schubert C; Doppler W; Grunicke H H
CORPORATE SOURCE: Institute of Medical Chemistry and Biochemistry, University of Innsbruck, Austria.
SOURCE: ADVANCES IN ENZYME REGULATION, (1994) 34 257-68.
Journal code: 0044263. ISSN: 0065-2571.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199411
ENTRY DATE: Entered STN: 19941222
Last Updated on STN: 20000303
Entered Medline: 19941115

AB HC-11 mouse mammary epithelial cells stably transfected with a glucocorticoid-**inducible** Ha-ras construct encoding a transforming (val12) p21Ha-ras were cotransfected with a c-fos-CAT construct containing the human c-fos **promoter** up to position -711 and the CAT **reporter gene**. Expression of Ha-ras by dexamethasone leads to a transcriptional activation of the fos-CAT construct which was found to be sensitive. . . PKC depletion following long-term exposure to TPA. The responsiveness to Ha-ras is retained if only the portion of the fos **promoter** covering the serum response element (SRE) and the adjacent fos AP-1 (FAP) site are put in front of a CAT **gene** linked to a thymidine kinase (TK) **promoter**. Further depletion of the FAP-site does not affect the inducibility by Ha-ras. Transcriptional activation of the SRE-FAP-TK-CAT as well as. . . blocked by long-term exposure to TPA. Long-term exposure to TPA depletes cells of PKC alpha and significantly reduces the PKC **epsilon** levels. Long-term exposure in bryostatin 1 selectively depletes PKC alpha. Depletion of PKC alpha by bryostatin 1 does not reduce. . . of the SRE-FAP-TK-CAT-construct by Ha-ras. It is concluded that (i) transforming Ha-ras induces c-fos in HC-11 cells via PKC (presumably **epsilon**), (ii) the signal is mediated to the serum response element (SRE) of the fos **promoter** and (iii) the fos AP-1 (FAP) site is not required for this mechanism.

L4 ANSWER 13 OF 13 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 92017835 MEDLINE
DOCUMENT NUMBER: 92017835 PubMed ID: 1922063
TITLE: Identification of a conserved lipopolysaccharide-plus-interleukin-4-responsive element located at the promoter of germ line epsilon transcripts.
AUTHOR: Rothman P; Li S C; Gorham B; Glimcher L; Alt F; Boothby M
CORPORATE SOURCE: Howard Hughes Medical Institute, Columbia University College of Physicians and Surgeons, New York, New York 10032.
CONTRACT NUMBER: AI-200057 (NIAID)
CA-40427 (NCI)
DK01336 (NIDDK)
+
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (1991 Nov) 11 (11) 5551-61.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199111
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19970203
Entered Medline: 19911114

AB . . . B-lineage cell lines with the mitogen lipopolysaccharide (LPS) and the lymphokine interleukin-4 (IL-4) induces expression of germ line

immunoglobulin C **epsilon** transcripts and class switching to the C **epsilon gene**. We show that LPS-plus-IL-4 induction of germ line **epsilon** transcripts (termed I **epsilon** transcripts) occurs at the transcriptional level in an Abelson murine leukemia virus-transformed pre-B-cell line. A 1.1-kb region of DNA surrounding the I **epsilon promoter** endows **inducible** transcription to a heterologous **reporter gene** stably transfected into these cells; such **inducible** expression depends on combined treatment with LPS and IL-4. Analyses of constructs transiently introduced into a B-cell lymphoma line demonstrated that LPS-plus-IL-4-**inducible** expression can be conferred by a 179-bp segment of DNA spanning the I **epsilon** transcriptional initiation site. Mutational analyses demonstrated that this expression depended on DNA sequences within a conserved region directly upstream from the I **epsilon** transcriptional initiation region. One nuclear protein that is constitutively expressed in normal B cells binds to the downstream end of. . . additional proteins, which are induced by IL-4 treatment of splenic B cells, bind to the transcription initiation sites of I **epsilon**. These proteins are indistinguishable in binding assays from proteins previously shown to bind an enhancer region of the class II major histocompatibility complex **gene** A alpha.